

Absence of epidemic outbreaks with heavy-tailed contact dynamics

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We study the epidemic spreading process following contact dynamics with heavy-tailed waiting time distributions. We show both analytically and numerically that the temporal heterogeneity of contact dynamics can significantly suppress the disease's transmissibility, hence the size of epidemic outbreak, obstructing the spreading process. Furthermore, when the temporal heterogeneity is strong enough, one obtains the vanishing transmissibility, hence the lack of epidemic outbreaks for any finite recovery time, the condition of which was derived.

Introduction— Throughout history, epidemics have had major influence not only on an individual's health but also on global human history [1], proving itself of great intellectual and practical interest [2–7]. The history of epidemics [1] shows that most of infectious diseases leading to pandemic state such as pest, cholera, malaria, and SARS (severe acute respiratory syndrome) were transmitted by broadcasting through carriers like animals and insects or media like polluted water or air without requiring direct physical contacts between susceptible and infected individuals. On the contrary, diseases requiring direct contacts for transmission such as sexually-transmitted diseases [8] have rarely been able to achieve pandemic states, with only a few contemporary examples such as AIDS.

Differences in transmission mechanisms for the two classes of infectious diseases may leave strong imprints on the characteristics of corresponding disease progression dynamics. For diseases transmitted through broadcast, explicit contact dynamics between individuals may become less important, and the classical theoretical approach based on the assumption of Poisson process and well-mixed population could reasonably be applicable [2–7]. Diseases requiring direct contacts for transmission, however, are directly governed by patterns of human activity, for a wide range of which from email exchanges to sexual contacts are known to exhibit heavy-tailed dynamics [9, 10], rendering the Poisson approach questionable [11].

Timings of individual's actions are typically highly heterogeneous. Human activity is dominated by a few bursts of activities, with extended periods of quiescence in between [9]. Such temporal heterogeneity [12] can be dictated by the waiting time distribution $P(\tau)$, where the waiting time τ is the time interval between two consecutive actions, and presents a new layer of complexity in social dynamics, parallel with the structural heterogeneity or network heterogeneity dictated by the complex network structure of contacts [13]. Indeed, it has been shown that such temporal heterogeneity can significantly affect to slow down spreading processes in networks [11, 14–18]. In this paper, we investigate further the effect of temporal heterogeneity of contact dynamics on the large-scale

properties of epidemics.

The main result of this paper is to show analytically that the temporal heterogeneity can significantly impede the spreading of epidemics, in stark contrast with the network heterogeneity that facilitates epidemic spreading [3]. Most dramatically, the epidemic outbreak can even be completely suppressed, given sufficiently strong temporal heterogeneity. We demonstrate this analytically by applying renewal theory [19, 20] to a prototypical epidemic model, the susceptible-infected-recovered (SIR) model. We derive expressions for the transmissibility and thus epidemic threshold for heavy-tailed $P(\tau)$, to show that the epidemic threshold increases with heterogeneity of contact dynamics without bound. The analytical predictions are well supported by extensive numerical simulations on random and scale-free networks. We conclude the paper by examining the effect of finite timescale and with discussion on the implications of our results.

SIR model with arbitrary contact dynamics— The SIR-type model we consider in the paper is formulated as follows. Each node is in one of three states, susceptible, infected, or recovered. The disease is transmitted through the contact between an infected node and its susceptible neighbor. In classical approach [2], the contact dynamics is assumed to be a Poisson process. Here we relax such Poisson assumption, and consider that the contact dynamics follows independent renewal process with a general inter-event time (waiting time) distribution $P(\tau)$. Along the way, the infected node can recover autonomously, after when it does not participate in epidemic dynamics. Classically the recovery dynamics is also assumed to be Poissonian. In this work we consider a fixed recovery time λ , to focus on the effect of heavy-tailed contact dynamics.

Key quantities for disease spreading dynamics are the so-called transmissibility T and the secondary reproductive number R [2, 4]. T is the probability that an infected individual would transmit disease to a susceptible neighbor before it recovers, and R is the expected number of secondary infections per each infected node. Given $P(\tau)$, λ controls the “infectiousness” of the disease (and thus T). If λ is large (small), there is more (less) chance for secondary infections. As the whole population becomes

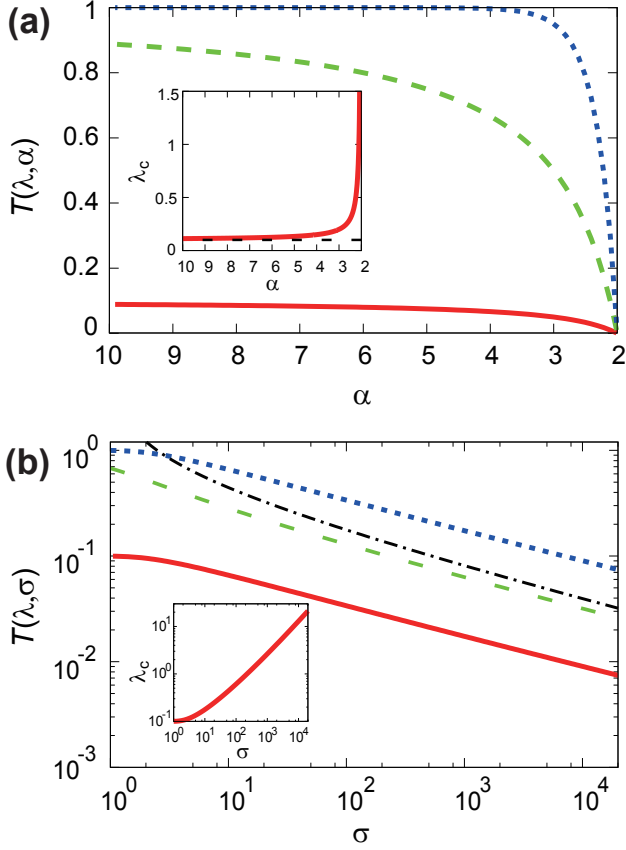


FIG. 1. Transmissibility T for (a) the power-law and (b) the lognormal $P(\tau)$ with $\lambda = 10^{-1}$ (solid), 10^0 (dashed), and 10^1 (dotted), plotted as a function of the power-law exponent α and the standard deviations σ of $P(\tau)$, respectively. $\tau_{\min} = 1$ in (a). The dash-dotted line in (b) denotes the asymptotic formula, Eq. (5), for the lognormal $P(\tau)$. (Insets) Epidemic threshold λ_c with $\kappa = 10$, vs. α (a) and σ (b).

composed of either susceptible or recovered nodes in the stationary state ($t \rightarrow \infty$), the average fraction of recovered nodes ρ_∞ in the stationary state measures the “size” of epidemic outbreak. One defines the epidemic threshold λ_c for the epidemic outbreak to be the infimum of λ such that $\rho_\infty > 0$.

Transmissibility and epidemic threshold— Following renewal theory [20], the transmissibility for contact dynamics following a renewal process with independent, identically distributed $P(\tau)$ and fixed recovery time λ can be obtained as

$$T = \int_0^\infty g(\Delta) \int_\Delta^\infty \delta(t_R - \lambda) dt_R d\Delta = 1 - \int_\lambda^\infty g(\Delta) d\Delta. \quad (1)$$

Here $g(\Delta)$ is the so-called generation time distribution [19], the distribution of time intervals between the moment of infection and the first following contact activity, which in this case is given by the residual waiting time distribution, $g(\Delta) = \frac{1}{\langle \tau \rangle} \int_\Delta^\infty P(\tau) d\tau$, where $\langle \tau \rangle$ is

the mean waiting time. The integral with respect to t_R accounts for the probability that the node does not recover during the interval Δ . The cases with general recovery time distribution $p(t_R)$ can be treated by replacing the delta function with $p(t_R)$ in Eq. (1).

In Fig. 1, we show the transmissibility T calculated for two heavy-tailed distributions that are widely used to model bursty dynamics [9, 21], (i) the power-law distribution with exponent α and minimum waiting time τ_0 ,

$$P_{PL}(\tau) = \frac{(\alpha - 1)}{\tau_0} \left(\frac{\tau}{\tau_0} \right)^{-\alpha} \quad (2)$$

for $\tau > \tau_0$ and $P(\tau) = 0$ otherwise, and (ii) the lognormal distribution with unit mean and variance σ^2 ,

$$P_{LN}(\tau) = \frac{1}{\tau \sqrt{2\pi \ln(1 + \sigma^2)}} \exp \left\{ -\frac{[\ln \tau + \frac{1}{2} \ln(1 + \sigma^2)]^2}{2 \ln(1 + \sigma^2)} \right\}. \quad (3)$$

In both cases, the transmissibility T decreases with the contact dynamics’ heterogeneity, dictated by either the power-law exponent α or the variance σ^2 of $P(\tau)$, and it even vanishes as α approaches to 2 (Fig. 1a) or as the variance diverges (Fig. 1b). This result clearly demonstrates that the heavy-tailed contact dynamics can drastically suppress, and even completely block, the epidemic spreading.

For a power-law $P(\tau)$, Eq. (2), the transmissibility can be calculated explicitly. It reads

$$T_{PL}(\lambda; \alpha) = \begin{cases} \frac{(\alpha-2)}{(\alpha-1)} \frac{\lambda}{\tau_{\min}}, & \lambda \leq \tau_{\min} \\ 1 - \frac{1}{\alpha-1} \left(\frac{\tau_{\min}}{\lambda} \right)^{\alpha-2}, & \lambda > \tau_{\min}. \end{cases} \quad (4)$$

Evidently, T_{PL} decreases with as α decreases and vanishes as $T_{PL} \sim (\alpha-2)$ as $\alpha \rightarrow 2$. For general $P(\tau)$, T may not always be obtained in a simple form. Yet its asymptotic behavior can be more accessible for many cases. For example, for the log-normal waiting time distribution, Eq. (3), the residual waiting time distribution is obtained as $g_{LN}(\Delta; \sigma) = \frac{1}{2} \left[1 + \operatorname{erf} \left(\frac{-\ln \Delta - \frac{1}{2} \ln(1 + \sigma^2)}{\sqrt{2 \ln(1 + \sigma^2)}} \right) \right]$, where $\operatorname{erf}(x)$ denotes the error function. Using properties of the error function, one obtains the leading asymptotic behavior of T for large σ as

$$T_{LN} \sim \sigma^{-1/4} / \sqrt{\ln(\sigma)}, \quad (5)$$

vanishing algebraically with σ (Fig. 1b).

Once T is obtained, the epidemic threshold can be readily obtained for the process on uncorrelated, tree-like networks, by mapping to a branching process [22]. From the criticality condition of the branching process, the condition for the epidemic outbreak is written as $R = T\kappa > 1$, where κ is the average branching number, given by the expected number of neighbors of an

infected node excluding the parent node. For an uncorrelated network κ is given by the expected remaining degree of a node reached by following a randomly chosen link, that is $\kappa = \sum_k (k-1)kP(k)/\langle k \rangle = (\langle k^2 \rangle - \langle k \rangle)/\langle k \rangle$. For power-law $P(\tau)$, Eq. (2), the epidemic threshold λ_c is explicitly obtained as

$$\lambda_{c,PL} = \begin{cases} \frac{\tau_{\min}(\alpha-1)}{\alpha-2} \frac{1}{\kappa} & (\lambda_c \leq \tau_{\min}), \\ \tau_{\min} \left[(\alpha-1) \left(1 - \frac{1}{\kappa}\right) \right]^{-1/(\alpha-2)} & (\lambda_c > \tau_{\min}). \end{cases} \quad (6)$$

λ_c increases and diverges as $\alpha \rightarrow 2$ (Fig. 1a, inset). Therefore, for $\alpha = 2$ the epidemic outbreak cannot take place for any finite λ , implying that only unrecoverable diseases ($\lambda = \infty$) can spread through the population. Similarly, λ_c diverges with σ for the lognormal $P(\tau)$ (Fig. 1b, inset). Finally, to obtain ρ_∞ , one can apply the generating-function method [4] based on the mapping to bond percolation in which each bond is randomly occupied with probability T .

Numerical simulations— We test the validity of the analytical predictions on two random network models, the Erdős-Rényi (ER) random graphs [23] and the static model of scale-free (SF) graphs [24]. The numerical simulation runs as follows. Initially all nodes are susceptible except for one infected node, chosen at random, as a seed node. Each connected pair of nodes makes contacts following a renewal process with the waiting time distribution $P(\tau)$, except for the first contact, the timing of which follows the residual waiting time distribution. Whenever an infected agent make a contact with a susceptible neighbor, the disease spreads, turning the susceptible node into infected. Along the way, each infected node recover after a fixed recovery period, λ . The process proceeds until there remains no infected agents in the network, and the final fraction of recovered nodes S is measured. The ensemble-averaged value of S over independent runs gives the expected outbreak size ρ_∞ .

On ER networks, we show the numerical simulation results with both the power-law and lognormal $P(\tau)$, together with the theoretical curves (Fig. 2a,b). The theoretical predictions are in excellent agreement with the numerical simulations. The epidemic outbreak size consistently decreases and the epidemic threshold diverges with the strength of temporal heterogeneity of contact dynamics, dictated by α approaching 2 (Fig. 2a) or diverging σ (Fig. 2b).

On SF networks with asymptotic power-law degree distribution $P_d(k) \sim k^{-\gamma}$, it is well-known that the epidemic spreading is facilitated to the extent that the epidemic threshold vanishes in the limit of infinite network size when $\gamma \leq 3$, as κ diverges with N [3]. To verify the impact of temporal heterogeneity in such a case, we perform numerical simulations with power-law $P(\tau)$ on the SF network with $\gamma = 2.5$ (Fig. 2c). The epidemic outbreak size decreases as α decreases, meaning that the temporal heterogeneity still hinders epidemic spreading in SF

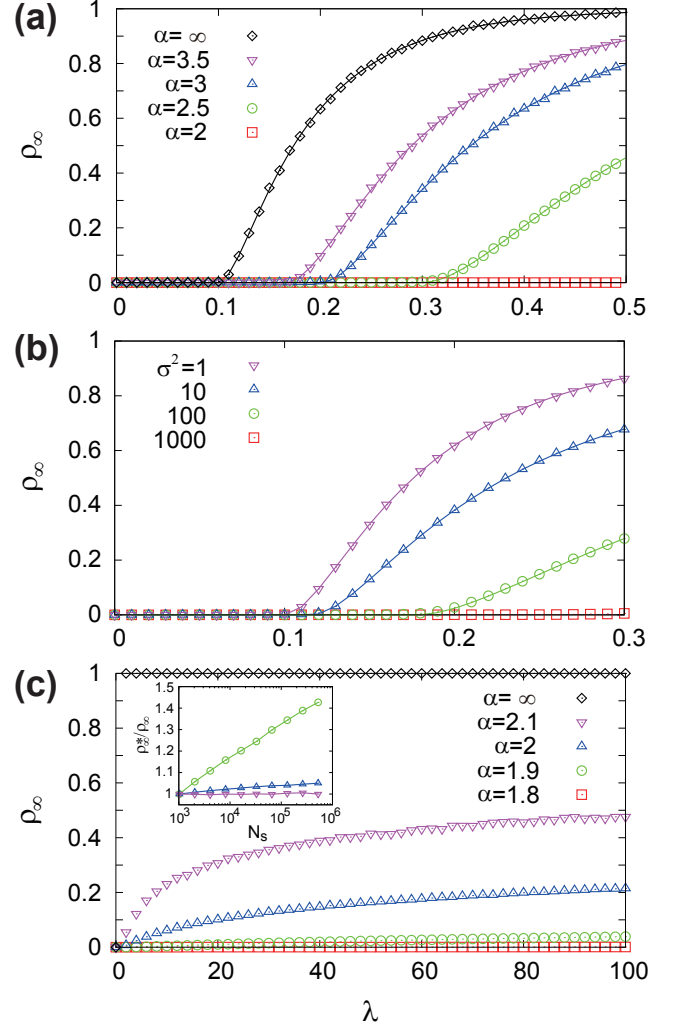


FIG. 2. Plots of the final density of recovered nodes (the expected outbreak size) ρ_∞ vs. the recovery time λ , of the SIR models. (a, b) Results on ER networks with mean degree $\langle k \rangle = 10$ and $N = 10^4$, for (a) the power-law and (b) the lognormal $P(\tau)$. (c) Results on SF networks with $\gamma = 2.5$ and $N = 10^4$ with the power-law $P(\tau)$. Symbols denote numerical simulation results and lines in (a, b) denotes the theoretical curves, in excellent agreement with each other. (Inset) Inverse of the numerically simulated ρ_∞ with $\lambda = 10$ (rescaled by its value ρ_∞^* for $N_s = 10^3$), plotted against the number of samplings of generation times N_s . For $\alpha = 2.1$ it remains constant, whereas it increases logarithmically for $\alpha = 2.0$ and 1.9 (same symbols as in main panel). Therefore, as $N_s \rightarrow \infty$, ρ_∞ is expected to vanish for $\alpha \leq 2$, as predicted by the theory.

networks. As long as $\alpha > 2$, however, $\lambda_c \approx 0$, that is, the epidemic outbreak occurs for any nonzero λ . In this sense, the network heterogeneity dominates over the temporal heterogeneity, when $\alpha > 2$. For $\alpha \leq 2$, however, the temporal heterogeneity can dominate over network heterogeneity, suppressing the epidemic outbreak completely even for SF networks. In numerical simulation, ρ_∞ is obtained to be nonzero, albeit small, for $\alpha = 2$

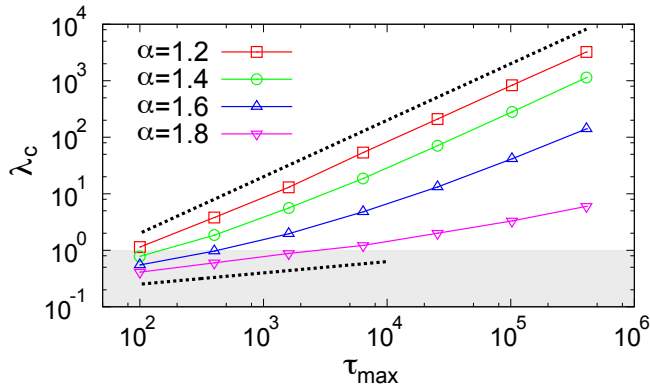


FIG. 3. The epidemic threshold λ_c vs. the maximum waiting time τ_{\max} in the power-law waiting time distribution with various exponent α , obtained from numerical simulations of the SIR model on the scale-free network with $\gamma = 2.5$ and $N = 10^4$. Dotted lines have slope 1.0 (top) and 0.2 (bottom), shown for comparison with the theoretical prediction, Eq. (8). Two regimes are separated by the minimum τ , $\tau_{\min} = 1$, indicated by the shade.

(and even for $\alpha = 1.9$), which is to be attributed to the finite number of samplings for the time to first contact from $g(\Delta)$ when $\alpha \leq 2$. Indeed, ρ_∞ is found to decay as the number of samplings is increased, and thus expected to vanish in the infinite-time limit even for SF networks, as predicted by the theory (Fig. 2c, inset).

Effect of the finite cutoff timescale— So far, we have assumed that there is no cutoff in the maximum waiting time in $P(\tau)$. In reality, however, contact dynamics mediating the spreading process takes place over a finite time window, bounded, for example, by an individual's lifespan. We examine the effect of such a cutoff timescale set by the maximum waiting time on the epidemic outbreak. With the cutoff waiting time τ_{\max} , the generation time distribution is given by $g(\Delta) = \frac{1}{\langle \tau \rangle} \int_{\Delta}^{\tau_{\max}} P(\tau) d\tau$. Let us take $P(\tau)$ to be a power law with exponent α in the range $(\tau_{\min}, \tau_{\max})$. For $\alpha > 2$, τ_{\max} plays only a minor effect in the transmissibility, neglectable for large τ_{\max} . Whereas for $1 < \alpha < 2$, reportedly corresponding to a number of human activities [10], τ_{\max} becomes the dominant term in $T(\lambda, \alpha)$ as

$$T(\lambda; \alpha) \sim \begin{cases} \lambda / \tau_{\max}^{(2-\alpha)} & (\lambda < \tau_{\min}), \\ (\lambda / \tau_{\max})^{2-\alpha} & (\lambda > \tau_{\min}). \end{cases} \quad (7)$$

Finally, the epidemic threshold λ_c depends on τ_{\max} as

$$\lambda_c \sim \begin{cases} \tau_{\max}^{2-\alpha} \lambda_{c,P} & (\lambda < \tau_{\min}), \\ \tau_{\max} \lambda_{c,P}^{1/(2-\alpha)} & (\lambda > \tau_{\min}). \end{cases} \quad (8)$$

where $\lambda_{c,P}$ denotes the epidemic threshold for Poisson contact dynamics (exponential $P(\tau)$). The predicted dependence of λ_c on τ_{\max} is well supported by the numerical simulations (Fig. 3). This result shows that the

more heavy-tailed (smaller α) the contact dynamics is, the larger is the impact of long but finite waiting times. In the $\tau_{\max} \rightarrow \infty$ limit, λ_c diverges with τ_{\max} , again completely suppressing the epidemic outbreak.

Summary— To summarize, we have shown both analytically and numerically that epidemic outbreaks of the SIR model can be strongly suppressed, and even completely blocked, by the heavy-tailed contact dynamics. Applying renewal theory, we have derived the transmissibility T and epidemic threshold λ_c for contact dynamics following power-law and lognormal waiting time distributions. It is shown explicitly that T vanishes (consequently, λ_c diverges) as $\alpha \rightarrow 2$ or $\sigma \rightarrow \infty$, respectively, which are specific instances of the general condition for diverging λ_c , given by $\int_{\lambda}^{\infty} g(\Delta) d\Delta = 1$ for any finite λ . As such, temporal heterogeneity is found to exert opposite effect to network heterogeneity, thus competing each other. Finally, it is noteworthy that although we have specifically formulated our analysis with the SIR dynamics with fixed recovery time, the main result of suppressing effect of temporal heterogeneity would apply under more general epidemic scenarios. For example, we observed similar effect for the SIR dynamics with distributed recovery times [25, 26] and the susceptible-infected-susceptible model [3, 6, 18].

As various kinds of human activities, including sexual activities [27], exhibit strongly heavy-tailed dynamics, our results should bear broad implications on epidemiological understanding and control of, for example, sexually transmitted diseases. Another interesting hypothesis that can be derived from our results is that the infectious diseases requiring direct physical contacts for transmission should necessarily have extremely long recovery time, were it to accomplish an epidemic outbreak. This may offer a Darwinian selection-based explanation on why such diseases as AIDS tend to be so resilient to cure that it takes excessively long for complete recovery, often persisting even throughout an individual's lifetime.

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